THE BIOLOGICAL SIGNIFICANCE OF PHYSIOLOGICAL HYPOXEMIA

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Preliminary inhalation of oxygen abolishes the rapid initial component of hyperventilation during physical work, which arises even before any change in the blood gases and, as has been suggested, is due to a reflex from receptors of the working muscles and the descending influence of the sensomotor cortex on the respiratory center [1, 5, 9, 17].

This effect is explained on the grounds that inhalation of oxygen suppresses the flow of impulses from the chemoreceptors, as a result of which the excitability of the respiratory center is lowered. Hence it can be postulated that the physiological hypoxemia, which exists under normal conditions, determines the level of excitability of the respiratory center which enables the rapid initial component of hyperventilation to take place during physical work and which prevents the fall in the partial pressure of oxygen in the arterial blood that would otherwise arise.

The view is widely held that intensification of activity of the respiratory muscles due to an increase in the resistance to respiration also arises through a reflex mechanism [3, 8, 15]. Preliminary inhalation of oxygen suppresses this response also. This last fact is an argument for rejection of its reflex nature and in favor of the view that it arises as a result of asphyxia [8, 13, 18].

In light of the above data the grounds for this conclusion are insufficient. Lowering of the excitability of the respiratory center due to hyperoxia may simply prevent the reflex action from the peripheral receptors on the respiratory center.

The results of the writers' experiments provide a wider view of the reflex mechanisms of the response to an increase in the resistance to respiration and they give a deeper insight into the biological role of the physiological hypoxia existing under normal conditions.

EXPERIMENTAL METHOD

Experiments were carried out on waking rabbits. Tracheotomy was performed on the animals under local anesthesia and a tracheotomy tube introduced into the trachea. Electrodes were sutured to the sternal part of the diaphragm. Complete obstruction of the respiratory passages for 5 or 6 inspirations was carried out before and after the rabbit inhaled pure oxygen for 1 min.

EXPERIMENTAL RESULTS AND DISCUSSION

While the animal inhaled air closure of the tracheotomy tube immediately induces a sharp increase in the first inspiratory volley. Subsequent volleys increased more and more and were converted into paroxysmal contractions of the diaphragm (Fig. 1A). Inhalation of oxygen for 1 min itself induced weakening and slowing of respiration. The explanation is that, as has already been stated, inhalation of oxygen inhibits the inspiratory activity of the respiratory center. Against the background obstruction of the respiratory passages led to a much smaller increase in the inspiratory volleys than in animals breathing air. Paroxysmal contractions of the diaphragm did not appear at all (Fig. 1B).

The reduction of the increase in intensity of inspiratory volleys arising in response to

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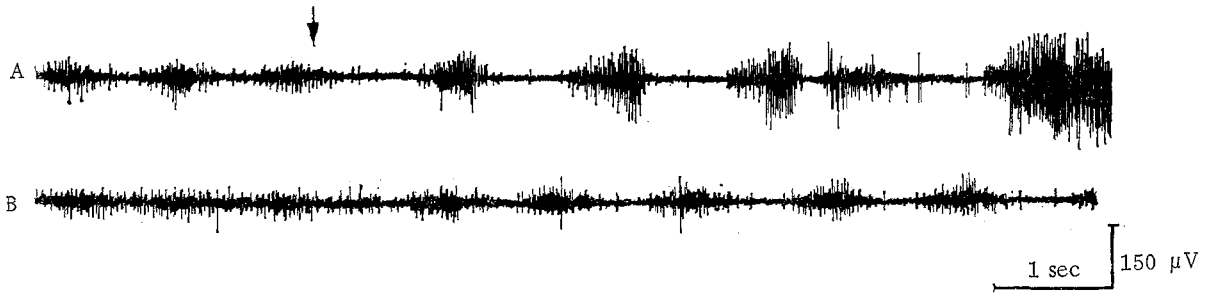

Fig. 1. Respiratory volleys of the diaphragm. Arrow indicates time of obstruction of tracheotomy tube. A) Before inhalation of oxygen; B) after preliminary inhalation of oxygen.

obstruction of the respiratory passages under normal conditions is evidence that lowering of the excitability of the respiratory center caused by hyperoxia prevents the respiratory center from responding and indicates the existence of humoral changes in the blood gases, developing during obstruction of the respiratory passages.

The fact deserves special attention that hyperoxia in these experiments prevented the onset of paroxysmal contractions of the diaphragm. The beginning of a sensation of respiratory discomfort during voluntary apnea is known to coincide with the beginning of paroxysmal contractions of the diaphragm [4]. In full agreement with this, some workers have found that during paralysis of the respiratory muscles the duration of apnea induced by stopping artificial respiration is increased, and no respiratory discomfort arises whatsoever [7, 11, 14]. Other workers, on the other hand, insist that cessation of artificial respiration gives rise to respiratory discomfort even when the respiratory muscles are paralyzed [2, 10, 12, 16]. In light of the present results it is an interesting fact that in experiments in which stopping artificial respiration in the presence of paralysis of the respiratory muscles did not give rise to respiratory discomfort the subjects were exposed to hyperoxia [7, 11, 14]. This is in harmony with the view that preliminary hyperoxia also increases the duration of voluntary apnea.

The normally existing physiological hypoxemia thus not only determines the level of excitability of the respiratory center, making possible a reflex increase in the activity of the respiratory muscles during physical work and when the resistance to respiration is increased, but also determines the onset, at the right time, of a sensation of respiratory discomfort, which leads to the resumption and voluntary strengthening of respiration.

Physiological hypoxemia is the result of imperfection of ventilation–perfusion relations, leading to incomplete aeration of the blood. The data given above are evidence that this "imperfection" in reality plays an important role in the adaptive activity of the body.

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